

Amendment Under 37 C.F.R. 1.116  
Capon et al.  
U.S. Serial No.: 09/321,589

II. In paragraph 7 on page 3 of the Office Action, the Examiner raised a new matter rejection relative to the claim language "which transduces a signal resulting in activation of a secondary messenger system in the absence of a T-cell receptor".

As discussed in the record and set forth in the instant specification, a receptor of interest signals in the absence of an intact T cell receptor. In any event, independent claims 57 and 59 do not include such language obviating the rejection.

III. On paragraph 8 on page 4 of the Office Action, claims 57, 64, 67 and 69 were rejected under 35U.S.C. §102(e) over Eshhar et al. (5,906,936).

The rejection is traversed for the following reasons. As the Examiner has acknowledged, Eshhar et al. describe chimeric receptor proteins which can use a T cell receptor alpha, beta, gamma or delta chain. Eshhar et al. do not describe receptors comprising a cytoplasmic domain taken from the CD3 zeta chain or the FcεR1 receptor, as encompassed by the current claims. Accordingly the rejection under 35U.S.C. §102(e) should be withdrawn.

IV. With regard to the observation that the previously filed Gorman et al. and Weiss references, in the file, were not properly made of record, a Form 1449 citing Gorman et al. and Weiss is submitted herewith for the convenience of the Examiner.

Amendment Under 37 C.F.R. 1.116  
Capon et al.  
U.S. Serial No.: 09/321,589

Marked-up version of the claims showing amendments. (08/238,405, filed 5 May 1994)

57. (Six times amended) A chimeric protein comprising in the N-terminal to C-terminal direction:

an extracellular antigen-binding domain of a single chain antibody that binds specifically to an antigen, wherein said antigen is a protein on the surface of a cell or a viral protein;

a transmembrane domain; and

a cytoplasmic domain which transduces a signal resulting in activation of a secondary messenger system [in the absence of a T-cell receptor, wherein said cytoplasmic domain is selected from the group consisting of] obtained from the CD3 zeta chain[, the CD3 eta chain, the CD3 gamma chain, the CD3 delta chain, the CD3 epsilon chain, the gamma chain of the F<sub>c</sub> receptor and a tyrosine kinase,] and wherein when said chimeric protein is expressed as a membrane bound protein in a selected mammalian host cell under conditions suitable for expression, said membrane bound protein initiates signaling in said host cell when said extracellular domain binds to said antigen.

59. (Twice amended) A chimeric protein [according to Claim 57], comprising in the N-terminal to C-terminal direction:

an extracellular antigen-binding domain of a single chain antibody that binds specifically to an antigen, wherein said antigen is a protein on the surface of a cell or a viral protein;

a transmembrane domain, and

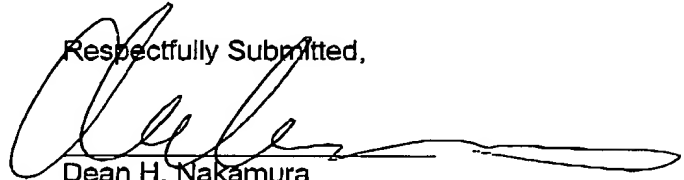
a cytoplasmic domain which transduces a signal [wherein the cytoplasmic domain is] obtained from the FcεR1 receptor, and wherein when said chimeric protein is expressed as a membrane bound protein in a selected mammalian host cell under conditions suitable for expression, said membrane bound protein initiates signaling in said host cell when said extracellular domain binds to said antigen.

Amendment Under 37 C.F.R. 1.116  
Capon et al.  
U.S. Serial No.: 09/321,589

CONCLUSION

The amendments set forth above place the claims in condition for allowance. Accordingly, an indication of allowance is respectfully requested. If any fees are necessitated by the filing of the Instant Amendment, the Commission is authorized to charge any such fees to Deposit Account No. 18-2220.

Respectfully Submitted,

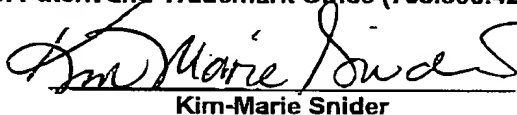


Dean H. Nakamura  
Attorney for Applicant  
Reg. No. 33,981

Roylance, Abrams, Berdo & Goodman  
1300 19<sup>th</sup> Street, N.W., Suite 600  
Washington, D.C. 20036-2680  
(202) 659-9076

Dated: 2 January 2002

I hereby certify that this Amendment (5 pages) and Form 1449 (1 page) were submitted by facsimile to the U.S. Patent and Trademark Office (703.308.4242) on 2 January 2002.

  
Kim-Marie Snider